

Historic, Archive Document

Do not assume content reflects current scientific knowledge, policies, or practices.

SF 607
us

Foreign Animal Disease Report

USDA
United States
Department of Agriculture
Animal and Plant
Health Inspection Service
Veterinary Services
MAR 10 '86

Emergency
Programs



Number 13-4

December 1985

In This Issue--

Vesicular Stomatitis Update
Puerto Rico Tick Program Update
Exotic Disease Agents in Animal Products
Nematodiriasis
Port Inspections
World Animal Disease Roundup
Babesiosis Review
Subject Index

Current Events

Vesicular Stomatitis Update

Cases of New Jersey type vesicular stomatitis (VS) were reported in Colorado starting in the Pueblo area on July 17, 1985 (see 13-3). This outbreak differed from the 1982-83 outbreak in that it did not spread statewide and remained primarily in the Pueblo and Durango areas. Two hundred and sixty-seven cases were investigated in Colorado. Of this total, 52 cases were confirmed by virus isolation and 94 cases were confirmed by serology. Sporadic cases were also reported near Albuquerque, New Mexico. In New Mexico, 89 cases were investigated, with 14 cases confirmed by virus isolation and 36 cases confirmed by serology. Arizona also reported their third case of VS in the northeast area of the State during the period July-September, 1985. (Dr. Wesley H. Garnett, 301 436-8091.)

Puerto Rico Tick Program Update

The tick eradication program for Boophilus microplus and Amblyomma variegatum in Puerto Rico continues to make substantial gains (see 13-2). A total of 12,222 tick-free herds were established by October 10, 1985.

Tick problems in Puerto Rico have been complicated by bovine babesiosis (cattle tick fever). (See article in September 1985 issue, 13-3.) By October 10, 1985, a total of 283 premises with approximately 20,684 heads of cattle were under quarantine for babesiosis or exposure to vector ticks. Of this total, 49 herds were diagnosed as infected with babesia based in part upon the results of laboratory examination of stained blood smears, and 118 herds were found positive to the babesiosis complement fixation test.

Pesticide treatment to destroy cattle ticks in infected herds is being done at 14-day intervals. (Dr. G. P. Combs, 301 436-8097.)

Exotic Disease Agents in Animal Products

The potential for an economic disaster caused by the introduction of exotic disease is a major concern of animal health personnel and others who are responsible for the protection of agricultural production. Foot-and-mouth disease (FMD), swine vesicular

disease (SVD), African swine fever (ASF), hog cholera (HC), and rinderpest (RP) are important diseases which have the potential to spread abroad through contaminated animal products.

In many countries, the import and export of animal products and biological products are strictly regulated. Requests for permits for the importation of animal products are individually evaluated for potential risk to domestic livestock.

A number of diseases of humans and lower animals have been transmitted by biological products. Examples are Creutzfeldt-Jakob disease transmitted through corneal transplants and the consumption of ethnic foods, FMD through smoked meats and vaccines, hepatitis through the consumption of shellfish, ASF through the feeding of contaminated meat scraps to swine, and SVD through the consumption of contaminated pork products.

Agent
Survival in
Animal Tissues

Agents of animal diseases vary in the length of time they will survive in animal tissues and secretions. For example, FMD virus survived up to 48 hours in skeletal muscle kept at 4°C. However, in lymph nodes, coagulated blood, and bone marrow obtained from the same carcasses, the virus persisted for at least 120 days at 4°C (Cottral, 1969). Microenvironments in these tissues protected the FMD virus from inactivation. The lower pH which accompanies other postmortem changes shortens virus survival time. Skeletal muscle pH values have been reported to be lower than those in lymph node tissues of the same carcass. (Garcia-Vidal, 1984).

High infectivity titers have been detected in the milk of lactating cows before the appearance of clinical FMD (Blackwell, 1975). The virus was also detected in bovine milk in the presence of antibody, 52 days after infection (Burrows, 1971).

Resistance to acid is greater with SVD virus than with FMD virus. Swine vesicular disease virus survived for 11 months in swine carcasses (Dawe, 1974).

African swine fever virus survived for 150 days at -4°C in swine carcasses and for 188 days at -4°C in bone marrow (Kowaleski, 1965). Rinderpest virus has been isolated from tissues more than a month after experimental infection and from carcasses buried for 2 months. Hog cholera virus survived in tissues for a period similar to that of RP virus. The viruses survived 33 days in skin, and 73 days in muscle (Gillespie, 1981).

The processing that is performed on meat and meat products to develop desirable flavors and prevent spoilage includes heat, salt concentration, and low pH, all of which adversely affect viruses. For example, FMD virus in infected lymph nodes packed in ground beef was inactivated by cooking in cans to a canning retort (cooker) temperature of 68.3°C. Also, FMD virus was inactivated in canned hams cooked to an internal temperature of 69°C (McKercher, 1978).

Thermal processing of meat products in flexible nylon tubes is milder than retort processing and, because of greater control of product quality, it has become popular among food processors. Foot-and-mouth disease virus survived in ground, infected skeletal muscle heated in flexible nylon tubes to an internal temperature of 71.5°C but did not survive a temperature of 79.4°C (Blackwell, 1984). In ground beef products containing milk from FMD-infected cows as a source of protein, and in products in which heart muscle was used as a supplement to skeletal muscle, FMD virus survived an internal temperature of 79.4°C (Blackwell, 1985).

In milk from FMD-infected dairy cows, the virus is readily detected before the onset of clinical FMD. The virus survived in milk after high temperature-short time (HTST) pasteurization to at least 71.7°C for at least 15 seconds, and HTST pasteurization in combination with evaporation (Blackwell, 1975). The virus survived HTST conditions of 75°C for 3 minutes, 85°C for 15 seconds (Blackwell, 1977), and 138°C for 3 seconds (Cunliffe, 1978).

Products such as cultured butter, cheese, non-fat dried skim milk, and other imported dairy products such as casein, sweet whey, beta-lactalbumin, and alpha-lactoglobulin have wide industrial use as extenders, simulated dairy products, emulsifying agents, ice cream mixes, candy mixes, stabilizers, paper coatings, adhesives, plastics, and man-made fibers. Foot-and-mouth disease virus survived the heat and acidic conditions of manufacturing cultured butter, precipitated casein, cheese, and sweet whey. However, the virus did not survive in acid whey, alpha-lactalbumin, beta-lactoglobulin, or lactose (Blackwell, 1984).

Processing of infected hams in cans to an internal temperature of 68.8°C was sufficient to inactivate SVD virus. However, the virus survived 400 days in dry salami and pepperoni sausage prepared by controlled acid fermentation, and in intestinal casings for over 2 years (McKercher, 1974). Soaking casings for 24 hours in 0.5% citric acid inactivated the virus.

Although neither ASF virus nor HC virus persisted longer than 30 days in fermented meat products, both viruses persisted for much longer periods of time in brined ham. Neither virus survived retort cooking to an internal temperature of 69°C (McKercher, 1978). In Prosciutto-type hams, SVD virus survived for 10 months (McKercher, 1985), and in salami, sausages and Serrano-type hams, ASF virus was not detected after 5 months (Ministry of Agriculture, Spain, 1982).

Detection of
Infectious Virus
Often
Difficult

Because of probable complexes formed between the progeny of infecting virus and host tissue constituents, the presence of viable virus is difficult to detect by cell culture infectivity assay (Blackwell, 1977). However, animal inoculation has proved consistently to be the most sensitive means to detect viruses. The demonstration of the formation of protective complexes between virus particles and tissue or food constituents complicates the testing of animal products for virus survival.

Experimental food products for virus survival tests should contain components from infected animals, rather than virus obtained from cell cultures or other artificial source.

Current Direction
in Food Processing
and Importing

New processing trends in the food industry and in consumer preferences dictate the development of manufacturing schemes that will produce the most bland product possible. These schemes include ultrafiltration, low temperature-long time heating, fermentation, and microwave heating. Although these processes are bactericidal for most contaminants, they favor virus survival.

Rapid worldwide distribution of animals and animal products has placed exceptional pressures on customs officials and port inspectors, and has diminished the effect of natural geographical barriers on the potential for the international spread of foreign animal disease. Also, the necessity to replenish an insufficient food supply in a country can cause an easing of import regulations that previously were stringently enforced. The risk of foreign animal disease introductions through animal products appears to be increasing.

Research
Needed

As international cooperative efforts have been initiated for the eradication of animal disease, so multiple disciplinary research efforts should be directed toward the development of food processing schemes that not only produce a satisfactory product but are also virucidal. (Dr. John H. Blackwell, Foreign Animal Disease Diagnostic Laboratory, NVSL, VS, APHIS, USDA; P.O. Box 848, Greenport, NY 11944; 516 323-2500.)

Nematodiriasis

Nematodiriasis is caused by infection by the parasitic nematode, Nematodirus battus, and other Nematodirus spp. in the small intestine of sheep. This disease is included in the list of diseases considered foreign to the United States (Proceedings 87th Annual Meeting, U.S. Animal Health Association, Las Vegas, Nevada, pages 11 to 23, 1983).

In February 1985, N. battus was identified from sheep in the Willamette Valley, western Oregon (Hoberg et al., in press, 1985). Until its discovery in Oregon, this parasite was known to occur only in Great Britain, Norway, the Netherlands, and Italy. Only the former two countries have reported clinical disease. Nematodiriasis is largely restricted to young lambs or weaner sheep and is not generally seen in sheep more than 3 or 4 months old, as older lambs and adult sheep develop immunity to the disease. However, these animals, in addition to young lambs surviving an initial infection, may carry small numbers of the parasite and therefore become important in the dissemination of eggs on pastures.

In northern Great Britain, where this nematode was originally described, N. battus is considered to be the most pathogenic parasite of young lambs. Clinical disease associated with N. battus has been known to affect up to 90 percent of lambs in some flocks, with losses approaching 30 percent.

The ability of N. battus to cause severe disease outbreaks in lambs is explained by its remarkable life cycle. Eggs laid by adult female worms in the small intestine of sheep are passed in the feces. Larval development to the infective third stage occurs entirely within the egg. Eggs of N. battus have tremendous capacity to survive freezing temperatures and have been known to remain viable on pastures for up to 2 years. This survival capacity of the egg assures continued passage among sheep from year to year. Generally, larvae will develop in eggs passed by infected sheep in the spring and then lie dormant through the remainder of the grazing season and winter. Warmer temperatures the following spring stimulate an abrupt and simultaneous hatch of third stage larvae. The simultaneous appearance of great numbers of infective N. battus larvae on herbage results in massive infective doses becoming available to lambs. Larvae of other species of Nematodirus that occur in sheep, such as N. spathiger and N. fillicollis, hatch in a more random manner.

Research in Great Britain and Norway has shown that N. battus must accumulate on a pasture over several years (usually 3) and coincide with the presence of susceptible lambs and a well-defined climatological regime for severe outbreaks to occur. The occurrence of clinical disease is determined by the time of larval hatch. For example, if larvae hatch too early (individual larvae are short-lived), lambs will not ingest enough of them on herbage to suffer a severe attack. On the other hand, if larvae hatch too late, the immunity of older animals limits the number of nematodes that develop. As a result, severe outbreaks of nematodiriasis do not always occur annually, but only in certain years when the correct factors coincide.

Most clinical infections occur in lambs 6 to 12 weeks old. Clinical signs include: 1. Acute enteritis accompanied by profuse diarrhea (diarrhea usually occurs 11 to 12 days post-infection); 2. Unthriftiness; 3. Rapid weight loss; and 4. Marked dehydration. In the most acute cases, death occurs within 2 to 3 days after the onset of diarrhea. Diarrhea associated with nematodiriasis may be confused with coccidial scours. However, blood is seldom seen in the feces with infections of N. battus. Most lambs in an affected flock will show clinical signs of similar severity within 6 to 10 days of the first appearance of disease. This is due to the simultaneous appearance of great numbers of infective third stage larvae.

The diagnosis of nematodiriasis is based on clinical signs and the identification of larval and adult worms in the small intestine. Fecal egg counts are apparently of little value since the disease occurs during the prepatent period of the parasite (before eggs are passed).

Nematodirus battus was not known to occur in the United States prior to February 1985. No specific anthelmintics have been developed for its control. Until such anthelmintics are developed and available, consideration should be given to the use of drugs which are registered for use against other species of Nematodirus.

Since its initial identification from sheep in Oregon, N. battus has been identified from two other flocks: One in the Willamette Valley and another along the Oregon coast. However, no death losses attributable to N. battus have been reported. (D. D. Wilson, Ph.D., Technical Support Staff, Emergency Programs, Veterinary Services, USDA, 301 436-8087, and Drs. G. L. Zimmerman and E. P. Hoberg, Oregon State University, 503 754-2927.)

Port Inspections

This article updates information given in 11-1, 1983, pages 9-11.

Travelers entering the United States at land, sea, and airports may be penalized if they attempt to smuggle prohibited plant and animal materials. The travelers are given at least two chances to declare to an inspector of the U.S. Customs Service (Customs) or U.S. Department of Agriculture (USDA) that they possess prohibited items. Those individuals caught with undeclared, prohibited animal or plant materials are immediately assessed a civil penalty. Penalties are paid directly to the Customs cashier, for the U.S. Treasury's General Fund. During fiscal year (FY) 1985, \$426,559 was collected from 14,966 "smugglers". Over 99 percent of the penalties were paid before travelers left the Customs area. Civil penalty procedures were implemented in 1983 to give the USDA Animal and Plant Health Inspection Service (APHIS) a new weapon against the introduction of foreign animal and plant diseases. Previously, travelers had little to lose when they failed to tell the Customs or USDA inspector about animal and plant products they were carrying. Only rarely did Customs assess a penalty.

Effective July 1, 1985, civil penalties of \$100 to \$750 are being assessed the owners of vessels which violate regulations for the safe disposal of ship garbage. A blue ribbon panel recommended in an August 1985 report to the Assistant Secretary for Marketing and Inspection that USDA should obtain authority for APHIS-Plant Protection and Quarantine (PPQ) to use all civil penalty funds to pay for specific public awareness activities.

From FY 1984 to 1985, there has been a 20 percent decrease in the numbers of civil penalties assessed. This suggests that civil penalties, coupled with a good public information program, are helping to reduce agricultural product smuggling.

Guidelines are being finalized for expansion of the PPQ detector dog program in which specially trained "sniffer" dogs help their human partners find illegal items. Teams are presently working at four international airports: John F. Kennedy International, Houston Intercontinental, Los Angeles International, and San Francisco International. Public acceptance of the tractable, trained beagles continues to be very favorable.

Even with improved public cooperation, APHIS is faced with a formidable challenge. Reports indicate that during the first 10 months of FY 1985, 121,591 noncommercial lots of meat and other animal products were confiscated at international airports and seaports, and from mail from foreign countries. (Dr. Robert Ormiston, 301 436-7633.)

Recent problems with **Foot-and-mouth disease (FMD)** in Italy appear to be subsiding. While as many as 100 new cases per month were reported during the height of the outbreak, there were only 18 cases in May, 5 cases in June, and 12 cases in August 1985. Elsewhere in Europe, no other outbreaks were reported during the current calendar year. In Africa, reports on FMD came from Tanzania and Cameroon. Most cases reported from Asia were caused by Type Asia₁, identified in Pakistan, Malaysia, and Thailand. Israel and Oman saw cases of Type O. In South America, the number of cases increased in Colombia, in the areas not covered by the United States-supported FMD program. The next highest number of cases was reported from Brazil, while elsewhere the incidence decreased.

Swine vesicular disease (SVD) has not been reported in over a year. A case of SVD has now been reported from Lower Saxony, West Germany, on or about October 14, 1985, in a herd of 520 swine. The herd was depopulated. Cases of SVD were found in France November 1983, and Italy August 1984. The last case in Germany was reported in July 1982.

The current world situation in regard to **African swine fever, (ASF)** can be summarized as follows:

The disease remains endemic on the African continent. However, so far in 1985, there have been no reports of new outbreaks.

The disease is considered to exist in Angola, Burundi, Cameroon, Malawi, Mozambique, Uganda, and Zaire. It probably exists in other areas of Africa without being reported. There are claims that it was eradicated from the Islands of Sao Tome' and Principe.

In Europe, ASF is reported from Spain and Sardinia. It invaded Belgium in early 1985, but eradication efforts appear to have been successful (see 13-3). The disease, at this time, does not exist on the Italian mainland, in France, nor on the island of Malta. Portugal is still considered affected by ASF.

In the Western Hemisphere, the disease made its first appearance in Cuba in the early seventies. It reappeared in the late seventies and was successfully eradicated from the Dominican Republic, Haiti, and Cuba. Brazil reported ASF for several years, beginning in 1978, but now claims the disease has been eradicated.

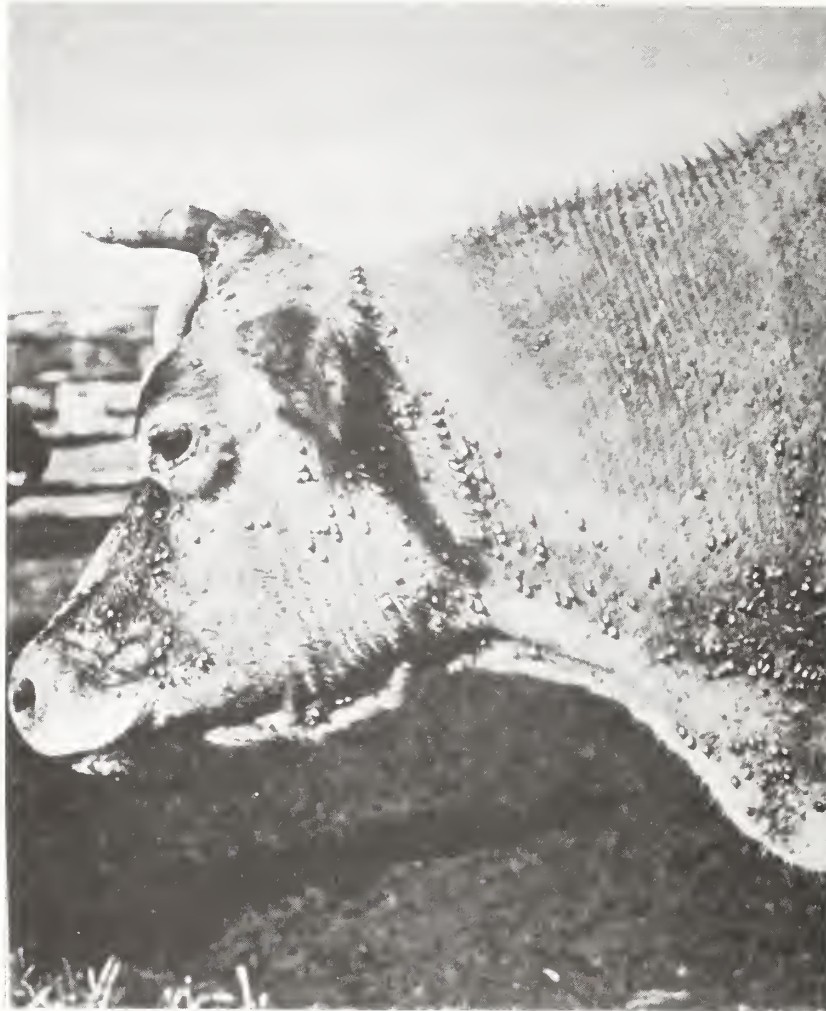
Rinderpest was again reported from several locations in Africa: Togo, Burkina Faso (Upper Volta), Ivory Coast, and Ghana. Of interest are unofficial reports that the disease may exist in North Korea.

Peste des petits ruminants had not been reported for quite some time, but an outbreak was noted in June in Oman. **African horse sickness**, another disease not heard from for a while, showed up in Ghana. Other diseases apparently only cropping up now and then are **sheep pox** and **goat pox**. They were recently reported in Israel and Morocco.

With available reports as indicators, **hog cholera** is showing some decrease worldwide, with most reported cases presently in Europe. (Dr. Hans J. Seyffert, 301 436-8285.)

Focus on...

Bovine Babesiosis



Taxonomic Classification

Two taxonomic classes of protozoan parasites are included in the subphylum Apicomplexa. The class Sporozoasida includes, among many others, the coccidial parasites, Eimeria, Plasmodium, and Toxoplasma. The class Piroplasmasida includes a single order, Piroplasmorida, and two families, Theileriidae and Babesiidae. The two genera, Theileria and Babesia, commonly referred to as piroplasms, are hemoproteozoans with many species parasitic to vertebrates. (Article on bovine theileriosis appeared in June 1985 issue: 13-2.)

History

Following the original description of Babesia bovis by Babes in 1888, Smith and Kilborne demonstrated the transmission of Babesia bigemina by Boophilus annulatus ticks in 1893. This was the first demonstration of parasite transmission by arthropods. Subsequent investigations firmly established ixodid (hard shelled) ticks as the principal vectors of cattle babesiae.

Hosts	<u>Babesia</u> species have successfully established themselves in various mammalian hosts, including all of the important domesticated animals, where at least two species, a "large" and "small," have been described. The table on the next page lists the six <u>Babesia</u> species of cattle, vectors, and geographic distribution.
Geographic Distribution	<p><u>Babesia bovis</u> is widely distributed in Boophilus tick vectors, especially in the tropics. Boophilus-transmitted <u>B. bovis</u> is considered a significant pathogen in Africa, southern Europe, Central and South America, and Australia. <u>Boophilus annulatus</u> is a <u>Babesia</u> vector in southern Europe and northern Africa. <u>B. microplus</u> is the principal vector in southern Africa, the Americas, and Australia. <u>Rhipicephalus bursa</u> has also been reported to transmit <u>B. bovis</u> in southern Europe and northern Africa. <u>Ixodes ricinus</u> has been described as a vector of this pathogen in Europe and, similarly, <u>I. persulcatus</u> in the USSR.</p> <p><u>Babesia bigemina</u> is transmitted by <u>B. microplus</u> ticks in Australia, and Central and South America. Cattle there are often infected with both <u>B. bigemina</u> and <u>B. bovis</u>. In Central America, <u>B. annulatus</u> is also involved. <u>B. annulatus</u> and <u>R. bursa</u> are vectors of <u>B. bigemina</u> in northern Africa. <u>B. decoloratus</u> and, apparently, <u>Rhipicephalus bursa</u> and <u>R. evertsi</u> also transmit <u>B. bigemina</u> in Southern Africa. <u>Haemaphysalis punctata</u> has been incriminated in <u>B. bigemina</u> transmission in Europe, and <u>Hyalomma anatolicum</u> transmits this parasite in India.</p> <p><u>Babesia major</u> is restricted to Europe. The vector there is <u>H. punctata</u>.</p> <p><u>Babesia divergens</u>, is transmitted by <u>H. punctata</u> and <u>I. ricinus</u>. <u>B. divergens</u> is the most economically important species in northern Europe.</p> <p><u>Babesia jakimovi</u> is a large species identified as separate from <u>B. bigemina</u> in 1977. It is transmitted by <u>I. ricinus</u> in Siberia. Its identity was confirmed by a lack of cross protection with <u>B. bigemina</u> and the fact that its host is the roe deer, which is refractory to <u>B. bigemina</u>. Because of the distribution of <u>I. ricinus</u>, further spread into Europe is a distinct possibility.</p> <p><u>Babesia occultans</u>, a large species, was recently isolated from <u>H. rufipes</u> ticks in South Africa. It is serologically different from <u>B. bigemina</u>, <u>B. major</u>, <u>B. bovis</u>, and <u>B. divergens</u>. <u>Babesia occultans</u> produces few visible parasites in the peripheral blood, and negligible clinical signs.</p>
Transmission	<p>After ticks acquire infection, a developmental cycle results in infective forms (sporozoites) within the salivary glands where they are released in saliva during feeding. Transmission can occur in three ways: 1. Stage-to-stage or transstadial, when both nymph and adult ticks transmit after having acquired infection in the previous stage, i.e., larvae or nymphs; 2. Transovarial, when female ticks acquire infection and pass it on through the eggs to the offspring where larvae transmit; and 3. Both transovarial and transstadial where larva, nymph and/or</p>

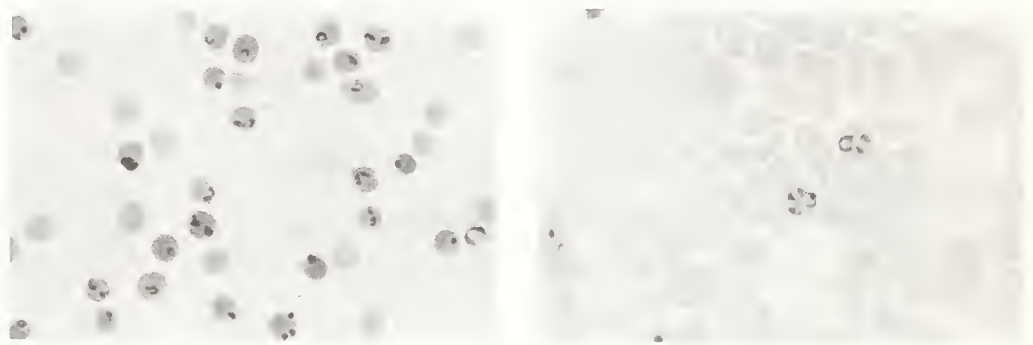
Bovine Babesia Species, Vectors and Distribution

<u>Species</u>	<u>Vector</u>	<u>Distribution</u>
<u>B. bovis</u> "small"	<u>Boophilus microplus</u>	Australia, South and Central America
	<u>B. annulatus</u>	Northern Africa, Southern Europe
	<u>B. decoloratus</u>	Southern Africa
	<u>Ixodes ricinus</u>	Europe and USSR
	<u>I. persulcatus</u>	USSR
	<u>Rhipicephalus bursa</u>	Europe and Northern Africa
<u>B. bigemina</u> "large"	<u>B. microplus</u>	Australia, South and Central America
	<u>B. annulatus</u>	Northern Africa, Central America
	<u>B. decoloratus</u>	Southern Africa
	<u>R. bursa</u>	Northern Africa
	<u>R. appendiculatus</u>	Southern Africa
	<u>R. evertsi</u>	
	<u>Haemaphysalis punctata</u>	Europe
	<u>Hyalomma anatolicum</u>	India
<u>B. major</u> "large"	<u>H. punctata</u>	Europe
<u>B. divergens</u> "small"	<u>H. punctata</u>	Europe
	<u>I. ricinus</u>	Europe
<u>B. jakimovi</u> "large"	<u>I. ricinus</u>	Siberia
<u>B. occultans</u> "large"	<u>Hyalomma rufipes</u>	South Africa

adult progeny transmit after initial infection of parental females.

Parasite
Development

There is no evidence which suggests that Babesia species of cattle infect any cell other than the erythrocyte. Infective forms or sporozoites are inoculated into the mammalian host during tick feeding. After penetrating the erythrocyte, the ring form or trophozoite appears. Parasite division results in two daughter merozoites. The merozoites represent the forms from which the term "large" and "small" derive. Merozoites emerge from parasitized erythrocytes and infect other erythrocytes.



The accompanying photographs show examples of a "large" (B. bigemina) and "small" (B. bovis) Babesia species in bovine erythrocytes.

Disease

Acute bovine babesiosis is often called "red-water" due to the common feature of hemoglobinuria. Other clinical signs include anorexia, dehydration, jaundice, and a febrile response which increases in parallel with the parasitemia. Direct damage to red cells and host response mechanisms result in intravascular hemolysis. Deaths result from pulmonary edema and anemic anoxia. If an animal survives the acute disease, it may remain a carrier for life. Subclinical infection usually protects the carrier animal from clinical babesiosis following further exposure. Clinical disease can recur if the balance between host and parasite is tipped in favor of the parasite due to stress, immunosuppression, or another disease.

Pathogenesis

The pathogenesis of anemia in babesiosis involves both damage to erythrocytes by escaping parasites, and antibody and complement-mediated removal of altered erythrocytes by the reticuloendothelial system. Histiocyte hyperplasia of the spleen, lymph nodes, and liver follows the onset of parasitemia. Circulating monocytes and neutrophils phagocytize damaged erythrocytes. The altered, infected cells may lyse. Lymphocytes and monocytes help establish protection, but also contribute to vascular damage and anemia.

In Babesia bovis and B. bigemina infections, and probably other Babesia spp. infections, the kallikrein system is activated by products released by the parasites and damaged tissues. Kallikreins are specialized enzymes found in blood plasma and other body substances. Their major action is the release of

peptides called "kinins" (e.g. bradykinin and kallidin). Kinins cause vasodilation, lower blood pressure, and increased vascular permeability, and they induce the contraction of smooth muscle. In babesiosis, the effects include edema, circulatory stasis, and shock.

In the liver, Kupffer's cells proliferate, impeding the flow of blood and resulting in congestion. Hepatic necrosis may occur.

Splenomegaly and enlarged lymph nodes are typically associated with the disease. Macrophages in the spleen and lymph nodes actively phagocytize infected erythrocytes. The kidneys may be swollen and dark in cases of acute B. bigemina infection, due to an inability to absorb the large amount of hemoglobin that has been released by hemolysis. Glomerulonephritis due to immune complexes has also been observed.

The pathological features just described may be seen in varying degrees of severity with all *Babesia* species afflicting cattle. However, in B. bovis infections the number of parasites in peripheral blood remains relatively low, while the central nervous system is often affected during the terminal stages of the acute disease. Parasitized erythrocytes appear to have a predilection for brain capillaries, where low oxygen tension prevails. Conditions required for continuous growth of B. bovis in vitro include low oxygen tension. Also, there appears to be an attraction of infected erythrocytes for endothelial tissue, where adherence of infected cells also contributes to the sludging of erythrocytes in brain capillaries. In vitro studies have shown that a proportion of B. bovis-infected erythrocytes adhere to endothelial cells.

Impacts on Agriculture

Babesiosis is especially important due to its wide geographic distribution and adverse effects on agricultural animal production. It is difficult to quantify losses due to babesiosis because more than one tick-borne disease often occurs in a given location. The lack of accurate diagnosis in many countries contributes to the incomplete epidemiological picture. In an effort to increase the availability of animal protein in developing countries, importation of cattle from nonendemic to endemic areas has increased. All too often, cattle are shipped to endemic areas without adequate methods to protect them from tick-borne diseases.

Babesiosis can cause death losses which may exceed 50% of susceptible cattle imported into an endemic area. Other costs include treatment, lost production, and control measures. In many developing countries, the cost is frequently too great to permit efforts to improve breeding stock for increased meat and milk production.

Diagnosis

Diagnosis of babesiosis depends on epidemiology, clinical signs, identification of the parasites in stained blood smears, and serology. Serological methods include precipitation and agglutination tests, complement fixation, fluorescent antibody tests, and enzyme-linked immunoassays. The indirect fluorescent antibody test has proven most reliable for the identification of

latent infections or carrier animals. Recent developments in enzyme immunoassays have focused on purified parasite antigens that are specific for Babesia species or strain.

Control

Control methods vary with the parasite and vector species, nature of the occurrence (epizootic or enzootic), and economic feasibility. Tick control, breeding of cattle for tick resistance, range management, chemotherapy, and immunoprophylaxis have lessened the severity of babesiosis in endemic areas and have reduced the potential for explosive epizootics.

Eradication of Babesia vectors has been successful only in the United States and a few islands. Subsequent growth of susceptible cattle populations has resulted in the need for strict tick surveillance programs along the Mexican border to ensure that Boophilus ticks are not reintroduced.

The breeding of tick resistant cattle has received increased attention in recent years. Bos indicus breeds have been shown to be more resistant to both ticks and Babesia than Bos taurus breeds. However, Bos indicus X Bos taurus crosses have generally been found to be as susceptible as Bos taurus breeds. An exception is the Australian milking zebu, a cross between high milk-yielding Jersey cows and relatively Boophilus tick-resistant Sahiwal (Bos indicus) bulls.

In established enzootic areas, calves are often naturally exposed to Babesia parasites while their innate resistance and protective maternal antibodies remain effective. Unfortunately, calves that fail to encounter parasites become susceptible later as adults. The key to enzootic stability lies in the maintenance of populations of infected ticks at levels adequate to induce premunition in all calves. Concurrent tick control with acaricides and pasture rotation must be carefully monitored to allow sufficient numbers of infected ticks to survive.

Babesiocides

Babesiocidal compounds are used both for treatment of acute disease and prophylaxis. The two most successful drugs are diminazene aceturate (Berenil) and imidocarb. Various treatment strategies have been investigated. In endemic areas, where most animals are naturally premunized, the drugs are administered to animals that develop an acute, life-threatening infection. The dose is adjusted so that the acute infection is resolved without eliminating the infection. This method allows for an immune response to develop along with the carrier state.

In non-endemic areas, or in cases where it is advantageous to sterilize the infection, Berenil usually has been effective. An exception is B. divergens, where some question exists concerning drug efficacy. Berenil is less effective than imidocarb as a prophylactic agent. Imidocarb has been used as a prophylactic agent in susceptible animals destined for movement to areas where exposure to Babesia is likely to occur and in other situations where Berenil is not effective. The concentration should be adequate for prevention of acute disease while allowing for sufficient multiplication of the parasites to ensure premunition. Inadequate dosage may result in fatal Babesiosis in some animals.

Higher dosages may prevent the development of the parasite altogether, leaving the animal susceptible to a later exposure.

Immunoprophylaxis

Immunoprophylaxis has been achieved either by the injection of dose-controlled virulent organisms followed by chemotherapy, or by the injection of attenuated organisms. However, several disadvantages accompany this approach: 1. It perpetuates the protozoan life cycle in the environment by establishing carrier animals; 2. There is variation in vaccine virulence, where attenuated parasites have occasionally reverted to full virulence and have caused mortality; 3. Some preparations have been contaminated with the agents of other blood-borne diseases, i.e., bovine leukosis virus; 4. The preparations are cumbersome, expensive to produce, and create storage, transportation, and handling problems; and 5. The preparations contain host erythrocyte stroma, which has resulted in neonatal isoerythrolysis in calves born to vaccinated dams.

Research

Experimental, inactivated vaccines and antigen preparations have produced resistance to homologous strains of Babesia. The protection that was produced was similar to the resistance that follows premunition. However, the vaccines were less effective than premunition against challenge with heterologous strains. Experimental vaccination with supernatant antigens from in vitro cultures of B. bovis produced partial protection that was not more effective than the protection which follows premunition. Experimental vaccines have not prevented the parasitemia which follows challenge with Babesia organisms.

Resistance to babesiacides and acaricides can be expected. Therefore, the development of a safe, effective vaccine for bovine babesiosis would be a major contribution to livestock production and development in endemic areas. Current research involves molecular biology and immunology, and focuses on the characterization and purification of molecular constituents of the parasites, together with a more thorough understanding of the bovine immune response, i.e., the immune mechanisms that operate during successful resolution of infection. (W. L. Goff, Ph.D., Hemoparasitic Disease Research Unit, Agricultural Research Service, U.S. Department of Agriculture, Pullman, WA 99164-6470.)

Subject Index

This subject index covers FAD Report volumes 10 through 13. It provides quick access to articles that contain information related to the index words. Readers who desire to maintain a complete file of the indexed articles can obtain copies of prior issues by sending a request to the editor. A subject index will be published each year in the winter issue.

Advisory committee	11(2):6-7	1983*
Africa, foot-and-mouth disease	13(1):6	1985
African swine fever:		
Belgium	13(2):11	1985
Belgium	13(3):6-8	1985
Cameroon	13(1):6	1985
Dominican Republic	10(1):2	1982
Haiti	10(2):3	1982
Haiti	10(3):2	1982
Haiti	11(1):3	1983
Haiti	11(2):2-3	1983
Haiti	11(3):2	1983
Haiti	11(4):2-3	1983
Haiti	12(4):1-2	1984
Malawi	13(1):6	1985
Potential impact,		
Canada	11(4):7-8	1983
Sardinia	10(2):9	1982
Sardinia	10(3):4	1982
African wild buffalo	13(2):12	1985
Alcelaphine herpesvirus-1	12(4):4	1984
Alcelaphine herpesvirus-2	12(4):4	1984
Alpacas imported from Chile	12(3):2	1984
<u>Amblyomma hebraeum</u>	12(3):2-3	1984
<u>Amblyomma species</u>	13(2):13	1985
<u>Amblyomma variegatum</u>	13(1):7	1985
Animal and Plant Health Inspection Service:		
Test exercise	10(2):1-2	1982
Test exercise	11(2):6	1983
Foreign Service	10(3):5-6	1982
Animal diseases eradicated from the		
United States	10(1):5-6	1982
Animal products, port inspections	11(1):9-11	1983
Animal products, exotic disease		
agents in	13(4):1-2	1985
Asia, foot-and-mouth disease	13(1):6	1985
Asiatic hemorrhagic septicemia:		
Pennsylvania, Texas	13(3):13	1985
review	13(2):6-11	1985
Audiovisuals, foreign animal diseases	13(3):9	1985

*Subjects are cited by volume number, (issue number): page number or span of pages, and year of publication.

Avian influenza:		
California	12(2):2	1984
Maryland	13(2):3	1985
Pennsylvania chickens	13(3):2	1985
Pennsylvania, Virginia	12(3):1	1984
Pennsylvania, Virginia	12(4):1	1984
Pennsylvania, Virginia		
New Jersey, Maryland	12(1):1-2	1984
Pennsylvania, Virginia		
New Jersey, Maryland	12(2):1	1984
Virginia turkeys	13(3):3	1985
Washington, DC	13(3):3	1985
Avian influenza economic assessment	13(1):1-3	1985
Avian influenza review	12(2):5-11	1984
Avian influenza surveillance completed	13(2):2	1985
Avian influenza update	13(1):1	1985
Avian influenza update	13(3):2	1985
Avian influenza virus:		
A/chicken/Pennsylvania/83	13(3):3	1985
in chicken eggs	13(1):3	1985
highly pathogenic	13(3):5	1985
H5N2	13(1):1	1985
H5N2 research	13(3):2-5	1985
H7N3	13(1):1	1985
H10N8	13(1):1	1985
in wildlife	12(2):2-4	1984
Babesiosis, cattle, Puerto Rico	13(3):1	1985
Babesiosis, horses, Puerto Rico	11(4):5	1983
Babesiosis review	13(4):8-14	1985
Belgium, African swine fever	13(2):11	1985
Benign African theileriosis	13(2):13	1985
Bird imports	10(1):1	1982
Bluetongue, Florida	11(4):3	1983
Bluetongue virus type 2	11(3):3	1983
Bovine theileriosis review	13(2):12-17	1985
Bureau of Animal Industry centennial	12(1):3	1984
Bureau of Animal Industry centennial	12(2):13	1984
Bureau of Animal Industry centennial	12(3):7	1984
Caliciviral disease review	11(3):8-16	1983
Cattle importation	12(1):4	1984
Cattle tick fever, Puerto Rico	13(3):1	1985
Central America and Panama		
veterinary services	11(3):3-7	1983
Central American animal diseases	11(3):6-7	1983
Contagious bovine pleuropneumonia		
review	12(1):6-8	1984
Contagious equine metritis	10(1):3	1982
Contagious equine metritis	10(2):4-5	1982
Contagious equine metritis	11(1):4	1983
Corridor disease	13(2):13-14	1985
Cosmopolitan theileriosis	13(2):13	1985
Dermatophilosis and heartwater,		
Caribbean	13(1):6-9	1985
Diptera, exotic	11(1):4-5	1983
Diptera, exotic	11(4):3-4	1983
Diseases eradicated from the		
United States	10(1):5-6	1982

Diseases present, Central America	11(3):6-7	1983
East Coast fever	13(2):13	1985
Economic assessment of avian influenza	13(1):1-3	1985
Editorial committee membership	12(1):8	1984
Editorial committee membership	12(4):15	1984
Embryo importation	11(2):4-5	1983
Emergency disease information	10(1):3	1982
Equine piroplasmosis, Puerto Rico	11(4):5	1983
Eradicated diseases, United States	10(1):5-6	1982
Exotic diptera	11(1):4-5	1983
Exotic diptera	11(4):3-4	1983
Exotic disease agents in animal products	13(4):1-2	1985
Exotic Newcastle disease (see Velogenic disease: VVND)	viscerotropic Newcastle	
Exotic ticks in Texas	12(3):2-3	1984
Food and Agriculture Organization	11(3):7-8	1983
Food Safety and Inspection Service	13(2):5	1985
Foot-and-mouth disease:		
in Africa	13(1):6	1985
in Asia	13(1):6	1985
in Chile	12(2):12	1984
in Colombia	11(1):5-9	1983
in Denmark	10(1):1	1982
in Denmark	10(2):2	1982
in Denmark	11(2):2	1983
in Elephants	12(4):6-7	1984
Geographic distribution	13(3):8	1985
in Italy	13(1):6	1985
in Italy	13(2):11	1985
in Italy	13(3):7-8	1985
in the Netherlands	12(1):3-4	1984
in South America	13(1):6	1985
in Mexico	13(1):10	1985
Foot-and-mouth disease vaccine bank	10(2):7-8	1982
Foot-and-mouth disease subunit vaccine	10(3):3	1982
Foot-and-mouth disease virus:		
effects of drying	13(2):5-6	1985
in animal products	13(4):1-2	1985
type Asia ₁	13(3):8	1985
survival, drying	13(2):5-6	1985
Foreign Animal Disease Advisory Committee	10(3):6-7	1982
Foreign Animal Disease Report:		
Editorial Committee	12(1):8	1984
Editorial Committee	12(4):15	1984
purpose	10(1):1	1982
Foreign disease investigations	13(1):5	1985
Foreign animal disease teachers seminar	10(3):7	1982
Foreign Service employment	10(2):8	1982
Foreign Service employment	10(3):5-6	1982
Gammaherpesvirinae	12(4):4	1984
Genetically engineered FMD vaccine	10(3):3	1982
Glanders, Turkey (see errata in 12-2, page 12)	11(4):5	1983
Guarding America's agriculture	11(1):9-11	1983
Haemaphysalis	13(2):13	1985

Harry S. Truman Animal Import Center Florida	10(3):4	1982
Haiti:		
African swine fever emergency	11(1):3	1983
African swine fever program	10(1):2	1982
African swine fever program	10(2):3	1982
African swine fever program	10(3):2	1982
African swine fever program	11(1):2-3	1983
African swine fever program	11(2):2-3	1983
African swine fever program	11(3):2-3	1983
African swine fever program	11(4):2-3	1983
African swine fever program	12(4):1-2	1984
Emergency declared	11(1):3	1983
No wild swine	10(1):2	1982
Heartwater:		
and dermatophilosis, Caribbean	13(1):6-9	1985
investigation	10(2):4	1982
review	10(1):6-10	1982
Hemorrhagic septicemia, Asiatic	13(2):6-11	1985
Hemorrhagic septicemia, Asiatic	13(3):13	1985
<u>Hippobosca longipennis</u>	11(4):3-4	1983
Hog Cholera:		
in Austria	11(3):16	1983
geographic distribution	11(1):4	1983
(see errata in 11-3, page 16)		
review	12(4):7-15	1984
How Foreign Animal Disease Report is produced	12(1):8	1984
<u>Hyalomma species</u>	13(2):13	1985
Importation of animals	12(3):2	1984
Imported cattle, Europe	12(1):4	1984
Imported pork	13(2):5	1985
Italy, foot-and-mouth disease	13(1):6	1985
Italy, foot-and-mouth disease	13(2):11	1985
Ivermectin	12(1):5	1984
Jembrana disease	13(3):10-13	1985
Llamas imported from Chile	12(3):2	1984
Los Angeles animal import center	12(3):2	1984
Malawi African swine fever	13(1):6	1985
Mali and Togo rinderpest	13(2):11-12	1985
Mali project	12(3):5	1984
Malignant catarrhal fever review	12(4):3-6	1984
Manila Office, Animal and Plant Health Inspection Service	12(4):3	1984
Maryland avian influenza	13(1):1	1985
Maryland avian influenza	13(2):3	1985
Mediterranean and tropical theileriosis	13(2):13	1985

Mexico foot-and-mouth disease survey	13(1):10	1985
Mexico screwworm program	12(4):2	1984
Mexico vesicular stomatitis	12(1):5	1984
Mexico vesicular stomatitis	12(3):4	1984
<u>Musca vitripennis</u>	10(2):2	1982
<u>Musca vitripennis</u>	11(1):5	1983
<u>Musca vitripennis</u>	11(4):3	1983
NADDS: National Animal Disease		
Detection System	13(2):3	1985
Nematodiriasis	13(4):4-6	1985
Newcastle disease in pet birds	13(1):5	1985
Newcastle disease in pigeons	13(1):5-6	1985
New animal import center in		
Los Angeles	12(3):2	1984
No wild swine in Haiti	10(1):2	1982
OIE: Office International des		
Epizooties	10(2):6-7	1982
One hundred years of animal health	12(2):13-14	1984
Oriental theileriosis	13(2):13	1985
Ossabaw Island, vesicular stomatitis on	11(4):1-2	1983
Panama and Central America	11(3):3-7	1983
Parafilaria in cattle, review	11(1):11-15	1983
Parafilaria, seasonal testing		
(see errata in 11-2, page 12)	11(1):15	1983
Parafilaria, therapy for	12(1):5	1984
Parafilaria vector	10(2):2	1982
Parent Committee: Import pathogens		
and vectors	13(1):9-10	1985
Penguin eggs imported	12(1):4-5	1984
Pennsylvania, avian influenza	13(1):1	1985
Pet birds, Newcastle disease	13(1):5	1985
Piroplasmiasis (see Babesiosis)		
Philippine scientific and technical		
exchange	11(2):5-6	1983
Plant Protection and Quarantine	11(1):9-11	1983
Plant Protection and Quarantine	13(4):6	1985
Pork imported	13(2):5	1985
Port inspections of animal products	11(1):9-11	1983
Port inspections of animal products	13(4):6	1985
Puerto Rico tick program	11(4):5-7	1983
Puerto Rico tick program	12(2):11-12	1984
Puerto Rico tick program	13(3):1	1985
Puerto Rico tick program	13(4):1	1985
Rama Dewa disease: Jembrana	13(3):10	1985
Rhinoceros, ticks in Texas	12(3):2-3	1984
Rift Valley fever, review	10(2):9-14	1982
<u>Rhipicephalus species</u>	13(2):13	1985
Rhipicephaline theileriosis	13(2):13	1985
Rinderpest in Africa	13(1):6	1985
Rinderpest, geographic distribution	13(3):9	1985
Rinderpest, Togo and Mali	13(2):11-12	1985
Rinderpest control in Africa	12(3):5-7	1984
Rinderpest review	11(4):8-12	1983
Rome office operations, Animal and		
Plant Health Inspection Service	12(4):3	1984
Sardinia, African swine fever	13(1):6	1985
Screwworm eradication in Mexico	12(4):2	1984

Screwworm program update	13(2):1-2	1985
Screwworm program review	11(2):7-11	1983
Sheep associated malignant catarrhal fever	12(4):4	1984
Sheep pox and goat pox geographic distribution	13(3):9	1985
SNOVET: Systematized Nomenclature of Veterinary Medicine	12(4):7	1984
Soft ticks on Hispanola Island	10(1):2	1982
South America foot-and-mouth disease	13(1):6	1985
Spanish language Foreign Animal Disease Report	12(4):7	1984
Survival of disease agents in animal products	13(4):2-3	1985
Suspected foreign animal diseases	10(2):4	1982
Suspected foreign animal diseases	11(1):4	1983
Suspected foreign animal diseases	11(2):4	1983
Suspected foreign animal diseases	11(3):3	1983
Tabanan disease: Jembrana	13(3):10	1985
Test exercise, Animal and Plant Health Inspection Service	10(2):1	1982
Test exercise, Animal and Plant Health Inspection Service	11(2):6	1983
Texas, exotic ticks	12(3):2-3	1984
Theileriosis, bovine, review	13(2):12-17	1985
Tick-borne protozoa	13(2):12	1985
Tick program in Puerto Rico	11(4):5-7	1983
Tick program in Puerto Rico	12(2):11-12	1984
Tick program in Puerto Rico	13(3):1	1985
Tick program in Puerto Rico	13(4):1	1985
Togo and Mali, rinderpest in	13(2):11-12	1985
Tropical theileriosis	13(2):13	1985
Truman, Harry S., Animal Import Center	10(3):4	1982
Velogenic vescerotropic Newcastle disease:		
in exotic birds	10(3):4-5	1982
geographic distribution in United States	10(2):4	1982
geographic distribution in United States	11(2):1	1983
geographic distribution in United States	11(3):1-2	1983
geographic distribution in United States	11(4):2	1983
geographic distribution in United States	12(3):1-2	1984
geographic distribution in United States	13(3):1	1985
Vesicular stomatitis conference	13(1):3-4	1985
Vesicular stomatitis in Missouri (see errata in 11-2, page 12)	11(1):1	1983
Vesicular stomatitis:		
geographic distribution in United States	10(3):1-2	1982
geographic distribution in United States	11(1):1	1983
geographic distribution in United States	11(2):1-2	1983

geographic distribution in United States	11(3):1	1983
geographic distribution in United States	12(2):11	1984
geographic distribution in United States	13(1):3	1985
geographic distribution in United States	13(3):2	1985
geographic distribution in United States	13(4):1	1985
historical review	10(3):11-14	1982
in Mexico	12(1):5	1984
in Mexico	12(3):4	1984
on Ossabaw Island, Georgia	11(4):1	1983
review	10(3):8-11	1982
vaccine	11(4):2	1983
Veterinary Services, Central America and Panama	11(3):3-7	1983
VVND: see velogenic viscerotropic Newcastle disease		
Wildebbeest-associated malignant catarrhal fever	12(4):4	1984
Wildlife avian influenza	12(2):2-4	1984
Wildlife disease studies	10(2):5-6	1982
World animal disease roundup	10(1):3-5	1982
World animal disease roundup	10(2):8-9	1982
World animal disease roundup	10(3):7	1982
World animal disease roundup	11(1):3	1983
World animal disease roundup	11(2):3-4	1983
World animal disease roundup	11(3):1-2	1983
World animal disease roundup	11(4):4-5	1983
World animal disease roundup	12(1):3-4	1984
World animal disease roundup	12(2):12	1984
World animal disease roundup	12(3):4	1984
World animal disease roundup	12(4):2-3	1984
World animal disease roundup	13(1):6	1985
World animal disease roundup	13(2):11-12	1985
World animal disease roundup	13(3):8-9	1985
World animal disease roundup	13(4):7-8	1985
Zimbabwean malignant catarrhal fever	13(2):13	1985

Questions about the FAD Report may be sent to:

Dr. E. I. Pilchard, Editor
APHIS, USDA, VS
Room 760, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

Thirty days before moving, send
address change, and if possible,
mailing label from latest issue to:

Information Management Branch
APHIS-USDA
Room G-187, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

☆U.S. GOVERNMENT PRINTING OFFICE: 1985-490-920-40007

